THE DIFFERENTIAL DIAGNOSIS OF ORGANIC FROM FUNCTIONAL DYSPEPSIA.*

By DR. DUDLEY FULTON, Los Angeles.

The method in vogue of studying the gastric functions, while advanced over those employed several years ago, are still inaccurate and deficient in diagnosing many cases of dyspepsia. Clinical instances are common with all of us in which, after a gastric analysis, we are still in doubt as to the pathological condition giving rise to the sub- or hyper-acidity, or whatever the stomach findings may happen to have been. One may easily quantitatively estimate the amount of acid or demonstrate the presence or the absence of ferments, but in pointing to the primary causes of these deviations from the normal, gastric analysis has decided limitations. In organic dyspepsia, the causes are to be found in anatomical alterations of some structure of the stomach wall. Ulcers, fissures, gastritis, cancer, dilatation and adhesions are the most common organic diseases. In functional dyspepsia, the digestive disturbances are dependent upon conditions remote from the stomach, and are not due to pathological alterations of it. Of the functional dyspepsias the most common and important are: the gastric neuroses, enteroptotic dyspepsia, the reflex dyspepsia caused by diseases of other organs—such as the gastric crises of tabes, and the dyspepsias due to chronic wasting disease—such as pernicious anæmia, etc.

The clinical differentiation of organic from functional dyspepsia is often very difficult, since the subjective symptoms and the laboratory findings are in many cases so nearly alike in both—especially if we accept the teaching of most of the standard works on the subject.

When properly diagnosticated, there are no internal diseases more satisfactorily treated than the dyspepsias.

It is beyond both the period allotted for the reading of this paper and my ability to cover the differential points between organic and functional dyspepsias. The following ideas, however, based upon liberal reading of the literature of gastric disorders and upon some 400 personal cases in which a test-meal was given to assist in making the diagnoses, may serve to bring out a discussion and the presentation of the views of others.

In our opinion more is to be learned of the dyspeptic by a careful history of his subjective symptoms and the physical examination, than by the test-meal. The latter is, however, indispensable in perhaps 25 per cent. of cases. Diagnoses based exclusively upon laboratory findings, are not often to be relied upon. The physiology and pathology of the gastric secretions, being modified as they are by psychical influences, body fatigue, by the quantity and quality of the food, as well as by the general state of health of the patient are too complex to be rightly interpreted by the findings of the test-tube and miscroscope of the laboratory alone. If, however, gastric analysis is used as a supplement to the clinical history and physical examination, it is of much value, particularly if several test-meals are given in the same case.

Cohnheim (1) of Berlin considers that in 75 per cent. of stomach cases a correct diagnosis can be made by the clinical history and the physical examination. The mistake most commonly made in examining stomach cases is, that a careful anamnesis is not obtained. Neither the questions of the physician nor the answers of the patient are precise, definite and accurate concerning subjective symptoms. Consequently enough facts are not obtained to assist materially in making the diagnosis, and the physician is as often led astray, as helped.

The history of a patient's suffering—the effect of food and the dependency, or independency of the symptoms upon the same; whether actual pain exists, or whether after closer questioning only pressure or fullness is said to be felt; whether the suffering is constant or only at certain regular, or irregular intervals after eating; whether pain is localized or diffuse or radiates to the sides or back; whether vomiting is a symptom, the time of vomiting in relation to meals, the character of the vomitus, etc., are of greater value in differentiating organic from functional dyspepsia than the test-meal.

We believe that when the correct interpretation of the subjective symptoms of a patient is given, and the physical examination carefully performed, the most important task in making a diagnosis of the majority of cases of stomach troubles has been fulfilled. It is well to always keep in mind that most of the cases of dyspepsia are functional.

The determination as to whether the patient has actual pain or only pressure or fullness is of the greatest practical importance, since pain is never found in functional dyspepsia. If actual pain exists, an organic lesion is present either in the stomach or in one of the neighboring organs. (2) We are well aware that in most of the text-books pain is considered a symptom of functional as well as of organic dyspepsia, that it occurs in simple hyperacidity and in the sensory neuroses of the stomach, and that neuralgia exists as a primary trouble, independent of lesions of the mucosa or other structures of the stomach. Theoretically speaking, gastralgia may exist as does trigeminal neuralgia. Practically, however, the term in its older sense is becoming obsolete. The operating table and autopsy findings are showing that gastralgia is almost always traceable to either the active lesions, or to the complications of the same, of the stomach or some neighboring organ. Less generally accepted is the view expressed by Leo, (3) Cohnheim and others, that the instances are very rare where pain is a symptom of an uncomplicated hyperacidity of the gastric juice. Lesions of the stomach mucosa: inflammation, ulcers, abrasions or adhesions are probably always present in those cases where real pain exists. We have found that in the management of these cases that if the treatment is

*Read at the Thirty-seventh Annual Meeting of the State Society, Del Monte, April, 1907.

based upon the assuption that all such are organic, good results are obtained, and we regard it a safe maxim to follow: when in doubt regard the condition as organic.

Functional dyspepsia, as mentioned above, rarely, if ever, produce actual pain. Upon close inquiry, such patients describe their discomforts in other terms, as pressure, fullness, heavy feeling, or are often unable to accurately describe their sensations. But they do not have real pain.

Ordinarily, a dyspeptic describes his discomforts by saying that he has "pain" no matter what his distress may be. It is always necessary to either support or to eliminate this expression, by further insistent questions, in order to learn anything by this very important quetsion. Furthermore, most patients state that the pain is in the stomach, whether it is in the epigastric region or in any other part of the belly.

We state, therefore, that as a working basis, actual pain in the epigastric region means organic disease of the stomach or of some neighboring organ.

Another differential point between the pain of organic, and the pressure or fullness of functional dyspepsia, is that the former is greatly modified and dependent upon the quality and quantity of the food, (4) while in functional stomach trouble, particularly nervous dyspepsia, the symptoms are aggravated more by excitement or general nervous irritability than by food. The symptoms occur after hard or soft foods, or with an empty or full stomach. In organic dyspepsia any diet which spares the diseased mucous membrane naturally modifies the amount of suffering. For instance, in gastritis, discomfort is felt only after eating hard foods (5) which mechanically irritate the mucosa, while in nervous dyspepsia, the pressure occurs after any kind of a diet. The distress and tenderness which is found on palpitation in nervous dyspepsia is diffuse: (6) that in ulcer circumscribed (7) and usually radiates (8) by preference around the left side to the 10th or 12th dorsal vertebra. (9) Increase of the pain, when the patient is in the right-sided position speaks for the location of the ulcer around the pylorus. Increase in the dorsal position, for the location on the posterior stomach wall. Increase in lying on the abdomen, for its location on the anterior stomach wall. (10) The fixity of the pain is also typical of ulcer (11). Stomach pains of a cramp or collicky character are due to involvement of one of the orifices of the stomach. The pain of cardia involvement usually occurs immediately or very shortly after eating. That of pyloric spasm three or four hours after eating. We regard spasm of the pylorus or cardia as always due to some organic lesion, and that neither occurs as an uncomplicated neurosis. The frequency of small breaks in the continuity of the gastric mucosa is now being rightly appreciated. These occur as minute ulcers, fissures, and erosions, easily overlooked during an operation or an autopsy. Just as these occur on the mucous membrane of the nose, mouth, lips, or anus, they occur in the stomach and are able to produce pain and distress which is usually wrongly attributed to hyperacidity or to a gastric neurosis. Characteristic of these small abrasions of the mucosa, is burning, boring pain three or four hours after eating, which if located within the sphincter area, assume a spasmodic character.

In discussing the differential diagnosis between atonic and mechanical conditions which give rise to dilatation of the stomach, a short review of the prevailing opinions on this subject is not out of place.

Most writers lay much importance upon atonic dilatation of the stomach. By this term they designate a relaxed, flaccid condition of the musculature which gives rise to much the same symptoms and disturbances as the dilatation due to other causes. The subject is one of the greatest practical importance, if we consider the term "atonic dilatation" as indicating an enlarged stomach together with inability to normally expel the food into the duodenum. This is Riegel's (12) conception of "dilatation" and corresponds to the term "ectasia" used by other writers. Every one agrees that any condition which prevents the food from passing into the duodenum within normal time limits, is a very serious condition. Disturbances of motility are of greater seriousness than disturbances of secretion. A stomach which empties normally may give rise to no symptoms—no matter what the quality of the gastric juice may be. On the other hand, a stomach which may be normal in all of its functions with the exception that stasis of its contents exists is always productive of serious gastric disturbances.

For diagnostic, practical purposes, no better means of diagnosising insufficient motility of the stomach has been suggested since Leube (13) advised washing the stomach out 6 or 7 hours after a full meal. If food is present, insufficiency of its motor powers is present. Since treatment of insufficiency must depend upon the cause of the stasis, the differential diagnostic principles are of the greatest practical importance in stomach work.

In the first place, the size of a stomach has nothing to do with its motor powers. A dilated stomach may empty normally, while food may remain within a normally sized stomach, hours longer than is physiological. From this it is clear that a dilated stomach is of no clinical importance so long as stasis of food is not an associated condition.

The important factor is food retention, not the size of the stomach. Dilatation is not a disease, but a symptom. If we exclude megalogastria and the acute dilatation following overloading the stomach and after abdominal operations, we believe that dilatation of the stomach is in by far the largest majority of cases due to mechanical obstruction, and that atonic conditions do not cause any serious food stasis. We regard every case in which after the administration of the test supper, remnants of it are found in the stomach the next morning, as due to organic obstruction of some sort.

We have never yet seen a serious case of food stasis due to atonic dilatation. If food stasis results from atonic conditions in which the musculature is too weak and flabby to expel its food into the

duodenum how are we to account for the fact that forced feeding improves the condition? If atonic dilatation existed, the introduction of more food would aggravate the condition as it aggravates the conditions in organic obstruction. The opposite is the case.

Faulty conclusions are often made by finding the stomach abnormally low, and by the occurrence of splashing sounds in the stomach. Neither are indicative of dilatation. In the first place the significance of splashing succusion sounds is due to more of the stomach surface being in contact with the stomach wall than is usual. Normally about one-third of the stomach is covered by the abdominal wall, the greater portion of the stomach being behind the liver and ribs. Any condition, therefore, which brings more of the stomach wall into immediate contact with the abdominal wall, such as congenital or acquired ptosis or dilatation, will permit splashing sounds to be easily produced. In the second place, the *sound* of splashing is of no consequence, but splashing which is *felt* is of practical value as indicating the lower border of the stomach. Splashing sounds do not have this significance.

The frequent diagnosis of dilatation is due to improper methods of investigation and wrong conclusions. Probably in the majority of the cases the condition is ptosis—a very common condition, while dilatation is by no means common. Persons with a congenitally narrowed thorax are predisposed to enteroptotic dyspepsia. Striller (14) of Budapest designates such persons as having the "habitus enteroptoticus." The long narrow thorax does not permit the stomach to occupy its normal position, but forces it into a more vertical position. More of the stomach wall is in contact with the abdominal wall and the lower border of the stomach reaches to the umbilicus or below it. This condition is mistaken very commonly for dilatation. Persons with the "habitus enteroptoticus" are predisposed to stomach troubles, dependent upon the malposition of the organ. Cohnheim has drawn attention to the very interesting fact that persons with this "habitus enteroptoticus" do not suffer from dyspeptic symptoms unless they are subnourished, and that the gastric disturbances are, as a rule, of a functional nature. We believe enteroptotic dyspepsia to be one of the most common forms of digestive trouble.

Perhaps none of the disturbances of the stomach require greater diagnostic judgment in differentiating organic from functional, than anomalies of secretion. It is such a fully accepted fact that hyper- and subacidity may be due to neuroses of secretions as well as in the various organic diseases, that after the gastric analysis has been made, one is still in doubt of the diagnosis and must usually depend upon the subjective symptoms and clinical examination for final judgment. In such cases several test-meals are usually required to be of any value. In those cases where there is no free HCl, one is able to eliminate organic disease if the gastric ferments are present in normal amounts. The mere quantitative demonstration of them will not suffice, as the ferments are present long after the HCl has disappeared. Boas' (15) method of measuring the functional activity of the lab ferment, as modified by Cohnheim (16) is so simple and effective that for practical work it is to be preferred to the more laborious quantitative estimation of the peptic activity, and since they run parallel, the measuring of one suffices for both. Only in interstitial gastritis and in atrophy of the mucosa are the ferments markedly depressed or absent, and the prognosis correspondingly bad.

Does hyperacidity exist as a primary condition, independent of organic lesions? We can safely say that at least such is the case only rarely. Hyperacidity is in by far the majority of cases secondary to (1) irritative conditions of the mucosa such as gastritis and ulcer, and (2) to those conditions which favor food stasis. The much discussed question as to whether hypersecretion is a symptom of food retention or due to a secretory neurosis is also still more or less unsettled, but we believe that both hyperacidity and hypersecretion are almost invariably dependent upon either irritative lesions or retention of food, and that we should be very cautious in making a diagnosis of nervous hyperacidity, or hyposecretion. Pure nervous hyperacidity is usually reflex from disease, usually organic, of some other abdominal or pelvic organ which of course requires the treatment rather than a stomach.

To summarize the views of this paper:

1. The importance of more accurate clinical investigation, and the inaccuracies of depending too fully upon "laboratory diagnosis."
2. Actual pain is always a symptom of organic disease of the stomach or of some neighboring organ.
3. The symptoms of organic dyspepsia are dependent upon quality and quantity of food.
4. These symptoms of nervous dyspepsia are independent of diet and vary with the degree of general nervous irritability.
5. Stasis of food within the stomach is probably never due to "atonic dilatation."
6. Hyper-acidity and hyper-secretion rarely occur independently of an organic lesion.

References:

1. Cohnheim; Die Krankheiten des Verdauungskanals, 1905.
2. Ibid.
3. Leo; Modern Clinical Medicine, p. 27.
4. Wegele; Therap. der Magen und Darmerkrank, p. 386.
5. Cohnheim; Ibid.
6. Mendel; Munch. Med. Wochenschr. No. 13.
7. Bourget; Die Krank. des Magens, 1906.
8. Schmidt; Die Schmerzphanomene 190!
9. Hemmeter; Diseases of the Stomach, p. 505.
10. Lorenz; Handbuch der Medizin, Ebstein, p. 127.
11. Rodari; Therap. der Magen, 1906.
12. Diseases of the Stomach, Nothnagel's Encyc. Med.

13. Leube; Med. Diagnosis.
14. Stiller; Berl. K. Wochenschr., Nos. 35, 39, 50.
15. Boas; Zeitschr. f. klin. Med., vol. 14.
16. Cohnheim; Ibid.

EFFECT OF CHEMICALS ON THE HEART NERVES.*

By WALTER E. GARREY, Ph.D., San Francisco.

I desire to present to-night a preliminary report of some experiments on the chemical stimulation of heart nerves. The work is, as yet, in its incipiency and not nearly so complete as I had hoped it would be when I consented to present it. The work is only an extension of work upon the chemical stimulation of nerves, so systematically done by Greutzner and A. P. Mathews for motor nerves, and by Greutzner for sensory nerves. So far as I have been able to discover, the cardiac nerves have not been worked on at all, and this investigation was undertaken in the hope of gaining new knowledge concerning the stimulating action of chemicals on nerves and of adding to our knowledge of the action of cardiac nerves. Especially was it hoped to segregate, by means of chemicals, different kinds of nerves running in mixed trunks. Thus far I have only succeeded in showing that efferent *cardiac* nerves may be stimulated by chemicals just as any motor or sensory nerve may be; only a limited number of chemicals, however, have been experimented with and the concentrations used have been few. The chemicals used have been those with which I have been led to expect the most pronounced stimulating effects, namely, solutions of those salts which precipitate or decrease the ionization of calcium or otherwise inactivate it, for it has been shown by Loeb and the author, in work, the complete report of which has not yet been published, that these are the salts, at least in their sodium combinations, which are most active in stimulating skeletal muscles and thereby inducing rhythmic twitchings.

Two possible effects of solutions must be borne in mind in any such investigation, viz.: the effects of concentration (osmotic pressure effects) and true chemical effects. These must be carefully segregated. For example, pure glycerin, as has been shown by Kuehne, is a powerful nerve excitant, but when diluted so that it is isotonic with the tissue and blood, has no such effect. Similarly the crystals or concentrated solutions of sodium chloride will quickly stimulate motor nerves and produce a tetanus of the muscle innervated while physiological salt solution has no such effect, or at least only after a very long latent period. The action of strong solutions, therefore, may be due to the extraction of water; in fact, Mathews has shown that almost any chemical, if the osmotic pressure of the solution is high enough (12 atmospheres), will give a primary stimulation of motor nerves, although the chemicals themselves may be powerful nerve depressants.

Osmotic Pressure Effects.—Most of my work has thus far been done on the vago-sympathetic nerve of the turtle, which is cut and the cardiac stump immersed in the solutions. In accord with the facts just stated, it is found that pure glycerin and concentrated solutions of sodium chloride or cane sugar or urea will effectually stimulate the cardiac nerves, producing both an augmentation of force of contraction, and slowing of the rate and finally bringing the heart to the diastolic standstill, while weaker solutions of the same chemicals, isotonic with the tissues, have no apparent action. The weakest solution of sodium chloride, which, when applied to the vagus nerve, has a marked effect upon the heart beat, is a half molecular solution ($\frac{m}{2} = 2.9\,\%$). This is four times the concentration of the blood or tissue.**

Chemical Effects.—The specific action of chemicals was tested by the use of $\frac{m}{8}$ solutions of the following salts: Sodium chloride, sodium phosphate, sodium carbonate, sodium citrate, sodium oxalate and sodium sulphate. With the exception of the first of these, all of the salts belong to the class of calcium inactivators referred to above, and in concentrations isotonic with the tissue, all of them, except sodium chloride, act as powerful nerve excitants, producing both an augmentation of the force and a decrease in the rate of the heart beat. The latent period in this form of nerve stimulation is relatively long, usually in the neighborhood of from two to ten minutes. But this latent period may be perceptibly shortened if the end of the nerve has been freshly cut. The first effect upon the heart is *always an increased height* of contraction, and this effect is also noted when concentrated solutions are used. The increased height may amount to treble the height of the ordinary contraction, and is evidenced in the contraction of both auricle and ventricle. Of all chemicals used, disodium phosphate seems to be most effective in this particular. The increased height is interpreted to mean a true augmentation of beat, and to be due to the stimulation of the augmentor fibres of the vago-sympathetic; this point, however, will be tested further. Following upon the augmentation there is a true inhibition of the rate which may be simply slowed or the contraction entirely inhibited. The inhibition is particularly well seen in one experiment with sodium citrate in which, after a latent period of two minutes, the heart was held in complete inhibition for forty-five minutes; it then beat for seven minutes, stopped again for seven minutes, beat for four minutes, stopped for three minutes and then took up an accelerated rate. The inhibition may involve the whole heart—i. e., sinus venosus, auricle, and ventricle. When the heart escapes from inhibition, the sinus invariably beats first,

* Abstract of a paper read before the Cooper Medical College Science Club, December 3, 1906.

** All concentrations are given in terms of molecular concentration, not in percentages, a necessity in comparative work on the action of the chemicals as has been pointed out by Greutzner and emphasized by Loeb.